

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re the Application of

Søren Mouritsen *et al.*

Serial No. 08/955,373

Filed: 21 October 1997

For: INDUCING ANTIBODY RESPONSE AGAINST SELF-PROTEINS WITH THE
AID OF FOREIGN T-CELL EPITOPES

Group Art Unit 1644

Examiner Schwadron

DECLARATION UNDER 37 CFR §1.132

Asst. Commissioner of Patents
Washington, D.C. 20231

Sir:

I, Dr Paul J Travers, hereby declare and state that:

1. I am Deputy Director of the Anthony Nolan Research Institute at the Royal Free Hospital in London. I hold a BA (Oxon) in the field of Biochemistry and a PhD (London) in the field of Genetics and Biometry. I have worked consistently in the field of immunology since 1979. I am *i.a.* co-author of the standard text book "Immunobiology – The Immune System in Health and Disease" published in 1999 by Current Biology Publications, part of Elsevier Science London. Other details relating to my scientific career appear from the attached Appendix A. I conclude that I am a skilled practitioner in the field of immunology. Moreover, I have since 1987 worked in the specialty of structural immunology, from 1987 to 1998 within the Department of Crystallography of Birkbeck College, London. I consider that I am knowledgeable also in the field of protein structure.
2. I have read and understood the disclosure in the above-captioned US patent application, including the presently pending claims. I have further studied and understood the Office Action dated 2 May 2000 in the above-captioned US patent application. I have also studied in detail the Russell-Jones *et al.* reference cited therein.
3. I have been asked by the Applicants in the above-captioned patent application to provide my comments to a number of issues raised in the Office Action dated 2 May 2000. In the following I provide these comments.
4. In paragraph 4 in the Office Action of 2 May 2000, the Examiner states that he finds the recitation of "essentially preserve the overall tertiary structure" unclear. It is my opinion as a skilled practitioner that this is not the case.

5. "Tertiary structure of a protein" is a term generally recognized in the arts of protein chemistry and immunology and relates to the 3-dimensional structure of a protein in its normal, folded state.
6. It is my opinion that the wording "essentially preserve overall tertiary structure" as used in the above-captioned patent application can be readily understood by the skilled reader. The specification points out on page 8, line 33, that the inventive technology disclosed in the patent application results in the induction of minimal tertiary structural changes of the native protein. Further, on page 10, lines 8-13, it is indicated that exercise of the technology of the patent application minimally obscures the tertiary structure of the self-proteins. Also, in the description on page 2, it is indicated that a problem with the known technique of coupling to carrier proteins is shielding of B-cell epitopes. These 3 passages in my opinion clearly indicate to the skilled reader that "essential preservation of overall tertiary structure" implies that when a peptide containing a T-cell epitope is substituted into a self-protein according to the above-captioned patent application, the substitution is one which introduces a minimum of disturbance in the tertiary structure of the self-protein whereby a maximum number of B-cell epitopes are preserved when comparing to the unmodified self-protein.
7. The Examiner seems to argue in paragraph 4 that preservation of immunogenicity implies preservation of overall tertiary structure. This argumentation is in my opinion not in line with basic facts in immunology and protein chemistry. It has been repeatedly demonstrated that a short peptide can be immunogenic, *i.e.* capable of inducing antibodies cross-reacting with a larger protein of which the protein fragment is part. Short peptides of *e.g.* 12 amino acids do not have any tertiary structure.
8. Therefore, "preservation of immunogenicity" is not the equivalent of "essential preservation of overall tertiary structure". Essential preservation of overall tertiary structure does in my opinion require that a large majority of B-cell epitopes in the native protein exists in the modified protein, whereas preservation of immunogenicity can be accomplished by even one single isolated B-cell epitope from an immunogen.
9. In paragraph 6 in the Office Action of 2 May 2000, the Examiner indicates that international patent application WO 92/05192, which is hereinafter designated Russell-Jones *et al.*, describes insertion and substitution of Trp peptides into immunogens so as to preserve overall tertiary structure and argues that the preservation of immunogenicity is the equivalent of preservation of tertiary structure. As detailed above in items 7 and 8, preservation of immunogenicity is not the equivalent of preservation of overall tertiary structure.
10. The Examiner also states in paragraph 6 of the Office Action that Example 5 in Russell-Jones *et al.* provides a teaching that would be applicable to any antigen. I do, as a person skilled in immunology, not agree with this interpretation of Example 5.
11. It is in my opinion clear that Example 5 exclusively relates to a substitution strategy which will and can be used in proteins containing suppressor regions. To the best of my knowledge, there are no mammalian self-proteins which contain such suppressor regions.

12. The specific vaccine construct suggested in Example 5 of Russell-Jones *et al.* has to the best of my knowledge never been reported tested in practice. Example 5 provides no demonstration that the suggested vaccine is effective in raising antibodies against gp120, *i.e.* the immunogenicity of the suggested construct is not verified. Absent any such evidence I would, as a skilled practitioner, not be inclined to use the suggested strategy on any other protein, let alone consider that the strategy would be useful in a protein lacking suppressor regions.
13. It is on the basis of the disclosure in Russell-Jones *et al.* impossible to determine whether the suggested vaccine construct in Example 5 1) will be immunogenic and give rise to antibodies against gp120 or 2) will preserve overall tertiary structure of gp120. The example provides no considerations which point to preservation of tertiary structure, but merely suggests that a suppressor region should be replaced with a Trat epitope.
14. Apart from Example 5, I can find no suggestions in Russell-Jones *et al.* to use a vaccine preparation strategy which involves substitution in any protein.
15. The Examiner also states that the gp120 variant of Example 5 "...would be no different the claimed invention..." even if suppressor regions had no effect on immunogenicity. As a person actively working in this field of technology, I cannot agree to this line of argumentation. Example 5 in Russell-Jones *et al.* suggests a specific strategy which is devised for proteins including suppressor regions and no suggestions are given to use the strategy in proteins without suppressor regions. The strategy in Example 5 relies on substitution of the suppressor region with an immunogenic Trat peptide. In contrast, the strategy of the above-captioned patent application relies on substitution of a region so as to preserve of tertiary structure. These two strategies do not seem to be able to result in the provision of identical vaccine constructs.
16. If relying on Example 5 in Russell-Jones *et al.* to prepare a vaccine against a self-protein, Example 5 would require that the skilled reader substitutes a suppressor region in the self-protein with a Trat epitope. This would not be possible, since self-proteins are not known to include suppressor regions. Example 5 further does not provide the skilled reader with any indication to make the insertion in a self-protein so as to preserve the tertiary structure.
17. In paragraph 6 of the Office Action, the Examiner implies that LH, somatostatin, inhibin, and FSH are disclosed in Russell-Jones *et al.* for use in a vaccine to be administered to the animal where these 4 molecules are autologous. I can find no indication in Russell-Jones *et al.* which states that LH, somatostatin, inhibin, or FSH should be used for immunization in an animal where these molecules are autologous. Russell-Jones *et al.* merely states on page 9, that vaccination with these 4 molecules is contemplated, but he does not indicate anywhere that the origin of the 4 molecules is the animal species to be vaccinated.
18. Russell-Jones *et al.* refers to LH, somatostatin, inhibin, and FSH as "immunogens". In the field of immunology, one would not usually describe a self-protein as an immunogen in the autologous host. In fact, the term "immunogen" is broadly defined as a "substance" which is capable of

inducing an immune response, and in their own right that is not the case for self-proteins in the autologous host. If using a strong adjuvant together with a self-protein in order to break autotolerance towards the self-protein, I would be inclined to use the term "immunogen" for the composition of self-protein and adjuvant and not for the isolated self-protein. This is because the "substance" which is capable of inducing antibody production against the self-protein is the composition of self-protein and adjuvant, whereas none of the components in isolation have this capability.

19. Russell-Jones *et al.* provides a broad statement that "...the immunogen is any molecule it is desirable to use to raise an immune response..." The Examiner interprets this sentence as an indication that the immunogens of interest according to Russell-Jones *et al.* are any molecules it could be of interest to immunize against. However, an at least equally meaningful interpretation of this sentence would be that the immunogen could be any type of known immunogen. Further, there is no indication that the immunogens according to Russell-Jones *et al.* would include non-immunogenic self-proteins in the autologous host. Russell-Jones *et al.* merely indicates in the paragraph bridging pages 8 and 9 that the Trat peptides are useful in weak immunogens as well as in strong immunogens, but it is not indicated that the technology would be effective in breaking autotolerance towards non-immunogenic self-proteins.
20. LH, somatostatin, inhibin and FSH have all previously been reported used as immunogens in animals where they are not "self". In such cases it has been necessary to use either strong adjuvants or carrier proteins because all 4 molecules, even when derived from other species than the one to be vaccinated, are weakly immunogenic.
21. It is therefore impossible to exclude that Russell-Jones *et al.* discloses an improvement in vaccination strategies involving xenogenic LH, somatostatin, inhibin, or FSH, and it is further impossible to conclude that Russell-Jones *et al.* discloses vaccination strategies involving proteins which are autologous to the vaccinated animal.
22. I have also been asked to comment on the results obtained in Example 4 of the above-captioned patent application.
23. From Example 4 and Fig. 5 in the above-captioned patent application it can be seen that the vaccination of mice with a murine TNF- α derived vaccine construct prepared according to the principles of the above-captioned patent application resulted in an earlier onset of detectable antibody production and a higher titre of anti TNF- α antibodies than did a traditional conjugate vaccine. Therefore Example 4 demonstrates that the technology disclosed in the above-captioned patent application provides superior results compared to known technology.
24. As a skilled practitioner in the field of immunology, I find the results reported in Example 4 and Fig. 5 surprising, since neither the earlier onset of the immune response nor the higher titre of the antibodies could be predicted on the basis of the knowledge available at the time of the invention.
25. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to

be true and further that these statements are made with the knowledge that wilful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such wilful false statements may jeopardize the validity of the application or any patent issuing thereon.

6 - Oct - 2000

Date

Paul Travers

Paul Travers

Appendix A: Curriculum Vitae, Paul J. Travers**CURRICULUM VITAE****PERSONAL INFORMATION**

Name: Travers, Paul Joseph

Born: 27 October 1956 Glasgow Scotland

Nationality: British

EDUCATION AND EXPERIENCE

1975-79 St Catherine's College, Oxford
BA (Hons) in Biochemistry (II.1)

Treasurer/Vice-President St Catherine's College Junior
Common Room 1976-77

Committee member OU Scientific Society 1977-78

Committee member OU Biochemical Society 1977-78

President OU Biochemical Society 1978-79

1979-83 Imperial Cancer Research Fund, London *and*
Dept of Genetics and Biometry, University College, London

PhD supervisors Dr W F Bodmer and Prof E B Robson

PhD awarded 1984 for thesis on "The expression of HLA-ABC and other
cell surface antigens by the trophoblast and tumour derived cell lines."

1984 Consultant, Intelligenetics Corp., Palo Alto, Ca.

1984-87 Research Fellow
Dept of Medical Microbiology
Stanford University Medical School

Cancer Research Institute Fellowship for studies on "The structure and
evolution of the MHC", with Prof H O McDevitt.

1987-98 Research Fellow
Dept of Crystallography
Birkbeck College, University of London

Royal Society 1983 University Research Fellowship for studies on "The
structure and function of MHC Class II antigens".

1999- Deputy Director, The Anthony Nolan Research Institute

FELLOWSHIPS AND AWARDS

- 1979 Imperial Cancer Research Fund Bursary
- 1984 Cancer Research Institute (NY) Fellowship
- 1985 NATO Travel Fellowship
- 1987 Royal Society 1983 University Research Fellowship
- 1987-1995 ICRF special project grant
- 1988 MRC project grant - Structure/function relationships of class II antigens
- 1993 SERC project grant - Isolation of cDNA and genomic clones coding for a diuretic peptide from *Acheta domesticus* (*with Dr K A Graeme-Cook and Dr G M Coast*)
- 1995 MRC project grant - Analysis of the interaction between T cell receptors and MHC:peptide complexes with agonist and antagonist peptides.
- 1999 Wellcome Project Grant Determination of the affinity of autoreactive TCRs and the role of CD8 (*with Dr A Cooke and Dr R Zamoyska*)

Honorary Positions and Appointments

- 1996 External Examiner, MSc in Biochemical and Medical Immunology, University of East London
- 1997 Honorary Senior Lecturer, Dept of Haematology, Royal Free Hospital School of Medicine
- 1998 Honorary Senior Lecturer, Dept of Crystallography, Birkbeck College
- 2000 Member, Subject Board in Pathology, University of London
- 2000 Chairman, Education Board,
British Society for Histocompatibility and Immunogenetics

PUBLICATIONS

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corresponding gene.
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Lee JS, Trowsdale J, Travers P and Bodmer WF
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phosphatase and other human trophoblast associated determinants.
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monoclonal antibodies to placental alkaline phosphatase.
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human tissue extracts using monoclonal antibodies in an enzyme immunoassay.
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Travers PJ, Blundell TJ, Sternberg MJE and Bodmer WF
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germ cell tumours evaluated by H17E2 monoclonal antibody assay
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Mitchison
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M. Perez-Rodriguez, J. R. Arguello, G. Fischer, S. T. Cox, E. Hossain, A. McWhinnie, P. J. Travers, S. G. E. Marsh and J. A. Madrigal
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Reviews

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